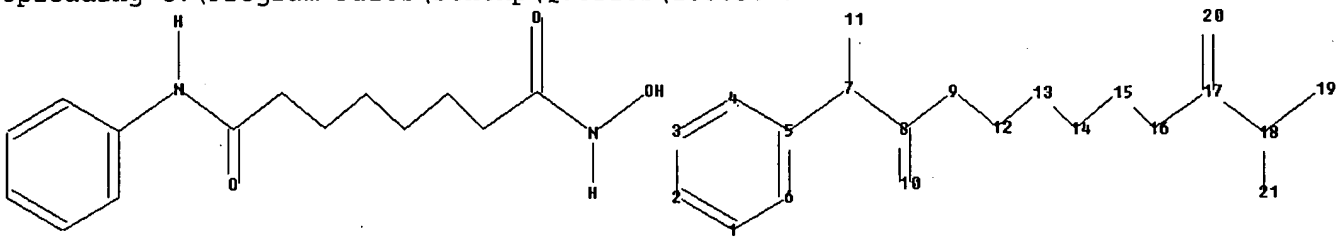
FILE 'HOME' ENTERED AT 12:18:16 ON 01 DEC 2006

=> **file registry**
=>
Uploading C:\Program Files\Stnexp\Queries\10665079.str

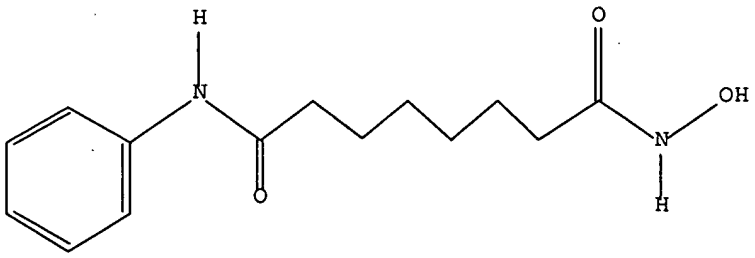


chain nodes :
7 8 9 10 11 12 13 14 15 16 17 18 19 20 21
ring nodes :
1 2 3 4 5 6
chain bonds :
5-7 7-8 7-11 8-9 8-10 9-12 12-13 13-14 14-15 15-16 16-17 17-18 17-20
18-19 18-21
ring bonds :
1-2 1-6 2-3 3-4 4-5 5-6
exact/norm bonds :
5-7 7-8 8-10 17-18 17-20 18-19
exact bonds :
7-11 8-9 9-12 12-13 13-14 14-15 15-16 16-17 18-21
normalized bonds :
1-2 1-6 2-3 3-4 4-5 5-6
isolated ring systems :
containing 1 :

Match level :
1:Atom 2:Atom 3:Atom 4:Atom 5:Atom 6:Atom 7:CLASS 8:CLASS 9:CLASS 10:CLASS
11:CLASS 12:CLASS 13:CLASS 14:CLASS 15:CLASS 16:CLASS 17:CLASS 18:CLASS
19:CLASS 20:CLASS
21:CLASS

L1 STRUCTURE UPLOADED

=> d l1
L1 HAS NO ANSWERS
L1 STR



Structure attributes must be viewed using STN Express query preparation.

=> **s l1**
SAMPLE SEARCH INITIATED 12:18:44 FILE 'REGISTRY'
SAMPLE SCREEN SEARCH COMPLETED - 175 TO ITERATE

100.0% PROCESSED 175 ITERATIONS 5 ANSWERS
SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS: ONLINE **COMPLETE**
BATCH **COMPLETE**
PROJECTED ITERATIONS: 2707 TO 4293
PROJECTED ANSWERS: 5 TO 234

L2 5 SEA SSS SAM L1

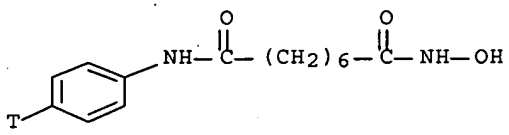
=> **s l1 exa full**
FULL SEARCH INITIATED 12:18:50 FILE 'REGISTRY'
FULL SCREEN SEARCH COMPLETED - 48 TO ITERATE

100.0% PROCESSED 48 ITERATIONS 2 ANSWERS
SEARCH TIME: 00.00.01

L3 2 SEA EXA FUL L1

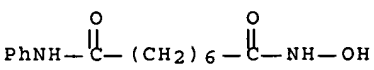
=> **d scan**

L3 2 ANSWERS REGISTRY COPYRIGHT 2006 ACS on STN
IN Octanediamide, N-hydroxy-N'-(phenyl-4-t)- (9CI)
MF C14 H19 N2 O3 T



HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):1

L3 2 ANSWERS REGISTRY COPYRIGHT 2006 ACS on STN
IN Octanediamide, N-hydroxy-N'-phenyl- (9CI)
MF C14 H20 N2 O3



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

ALL ANSWERS HAVE BEEN SCANNED

=> **file medline, caplus, uspatfull, wpids**

=> **s l3**
SAMPLE SEARCH INITIATED 12:19:26 FILE 'WPIDS'
SAMPLE SCREEN SEARCH COMPLETED - 15 TO ITERATE

100.0% PROCESSED 15 ITERATIONS 4 ANSWERS
SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS: ONLINE **COMPLETE**
BATCH **COMPLETE**
PROJECTED ITERATIONS: 34 TO 266
PROJECTED ANSWERS: 4 TO 99

L4 378 L3

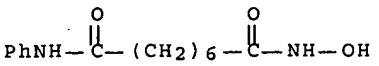
=> **s l4 and "b-cell lymphoma"**
L5 10 L4 AND "B-CELL LYMPHOMA"

=> **d l5 1-10 ibib, abs, hitstr**

L5 ANSWER 1 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2006:909614 CAPLUS Full-text
DOCUMENT NUMBER: 145:363490
TITLE: The medicinal composite for treating B cell lymphoma
INVENTOR(S): Chen, Zhu; Chen, Saijuan; Zhao, Weili; Wang, Lan; Liu, Yuanhua; Yan, Jinsong
PATENT ASSIGNEE(S): Ruijin Hospital Affiliated To Shanghai Jiaotong University School of Medicine, Peop. Rep. China
SOURCE: Faming Zhuanli Shenqing Gongkai Shuomingshu, 20pp.
CODEN: CNXXEV
DOCUMENT TYPE: Patent
LANGUAGE: Chinese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CN 1824307	A	20060830	CN 2005-10112147	20051228
PRIORITY APPLN. INFO.:			CN 2005-10112147	20051228

AB This invention relates to a medicinal composite for treating B cell lymphoma, which comprises Rituximab 1-100 μg/mL, SAHA (suberoyl anilide hydroxamic acid) 1-10 μM. Title has obvious synergistic action of inhibition of B lymphoma cell growth and breeding, synergistic inducement of apoptosis, synergistic inhibition of NF-κ B activity and lowing BCL-XL expression.
IT 149647-78-9, Suberoyl anilide hydroxamic acid
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(medicinal composite for treating B cell lymphadenopathy)
RN 149647-78-9 CAPLUS
CN Octanediamide, N-hydroxy-N'-phenyl- (9CI) (CA INDEX NAME)

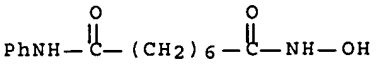


L5 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2006:459141 CAPLUS Full-text
DOCUMENT NUMBER: 144:460489
TITLE: Histone Deacetylase Inhibitors Suppress the Inducibility of Nuclear Factor-κB by Tumor Necrosis Factor-α Receptor-1 Down-regulation
AUTHOR(S): Imre, Gabriele; Gekeler, Volker; Leja, Astrid; Beckers, Thomas; Boehm, Markus
CORPORATE SOURCE: Therapeutic Area Oncology, ALTANA Pharma AG, Konstanz, D-78367, Germany
SOURCE: Cancer Research (2006), 66(10), 5409-5418
CODEN: CNREA8; ISSN: 0008-5472
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Recently, the inhibition of histone deacetylase (HDAC) enzymes has attracted attention in the oncol. community as a new therapeutic opportunity for hematol. and solid tumors including non-small cell lung cancer (NSCLC). In hematol. malignancies, such as diffuse large B -cell lymphoma, the HDAC inhibitor (HDI), suberoylanilide hydroxamic acid (SAHA), has recently entered phase II and III clin. trials. To further advance our understanding of their action on tumor cells, we investigated the possible effect of HDI treatment on the functionality of the nuclear factor-κB (NF-κB) pathway in NSCLC. We found that in the NSCLC cell lines, A549 and NCI-H460, the NF-κB pathway was strongly inducible, for example, by stimulation with tumor necrosis factor-α (TNF-α). Incubation of several NSCLC cell lines with HDIs resulted in greatly reduced gene expression of TNF-α receptor-1. HDI-treated A549 and NCI-H460 cells down-regulated TNF-α receptor-1 mRNA and protein levels as well as surface exposure, and consequently responded to TNF-α treatment with reduced IKK phosphorylation and activation, delayed IκB-α phosphorylation, and attenuated NF-κB nuclear translocation and DNA binding. Accordingly, stimulation of NF-κB target gene expression by TNF-α was strongly decreased. In addition, we observed that SAHA displayed antitumor efficacy in vivo against A549 xenografts grown on nude mice. HDIs, therefore, might beneficially contribute to tumor treatment, possibly by reducing the responsiveness of tumor cells to the TNF-α-mediated activation of the NF-κB pathway. These findings also hint at a possible use of HDIs in inflammatory diseases, which are associated with the overprodn. of TNF-α, such as rheumatoid arthritis or Crohn's disease.

IT 149647-78-9, Suberoylanilide hydroxamic acid
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(histone deacetylase inhibitors suppress the inducibility of nuclear factor-κB by tumor necrosis factor-α receptor-1 down-regulation)

RN 149647-78-9 CAPLUS
CN Octanediamide, N-hydroxy-N'-phenyl- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 3 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2006:82439 CAPLUS Full-text
DOCUMENT NUMBER: 144:403895
TITLE: Clinical experience with intravenous and oral formulations of the novel histone deacetylase inhibitor suberoylanilide hydroxamic acid in patients with advanced hematologic malignancies
AUTHOR(S): O'Connor, Owen A.; Heaney, Mark L.; Schwartz, Lawrence; Richardson, Stacie; Willim, Robert; MacGregor-Cortelli, Barbara; Curly, Tracey; Moskowitz, Craig; Portlock, Carol; Horwitz, Steven; Zelenetz, Andrew D.; Frankel, Stanley; Richon, Victoria; Marks, Paul; Kelly, William K.
CORPORATE SOURCE: Department of Medicine, Division of Hematologic Oncology, Lymphoma Service, Leukemia Service, Division of Solid Tumor Oncology, Developmental Chemotherapy Service, Genitourinary Oncology Service, Department of Radiology, Department of Nursing, Cell Biology Program, Mem. Sloan-Kettering Cancer Center, Sloan-Kettering Institute, New York, NY, USA
SOURCE: Journal of Clinical Oncology (2006), 24(1), 166-173
CODEN: JCONDN; ISSN: 0732-183X
PUBLISHER: American Society of Clinical Oncology
DOCUMENT TYPE: Journal
LANGUAGE: English

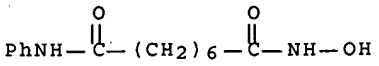
AB Purpose: To document the toxicity and activity of the histone deacetylase inhibitor suberoylanilide hydroxamic acid (SAHA) in patients with pretreated hematol. malignancies. Patients and Methods: Two formulations of SAHA (i.v. [IV] and oral) have been assessed in two consecutive phase I trials. In both trials, dose escalation was performed in parallel and independently in patients with solid tumors and hematol. malignancies. Eligible patients were required to have adequate hepatic and renal function, an absolute neutrophil count ≥ 500/μL and a platelet count more than 25,000/mL. All patients provided informed consent for study inclusion. Results: A total of 39 patients with hematol. malignancy were enrolled (14 on IV SAHA and 25 on oral SAHA), of whom 35 were treated. The spectrum of diseases included patients with diffuse large B- cell lymphoma (n = 12), Hodgkin's disease (HD; n = 12), multiple myeloma (n = 2), T-cell lymphoma (n = 3), mantle cell lymphoma (n = 2), small lymphocytic lymphoma (n = 2), and myeloid leukemia (n = 2). Major adverse events with the oral formulation included fatigue, diarrhea, anorexia, and dehydration, whereas myelosuppression and thrombocytopenia were more prominent with the IV formulation. Typically, the hematol. toxicities resolved shortly after SAHA was stopped. There was no neutropenic fever or neutropenic sepsis. Reduction in measurable tumor was observed in five patients. One patient with transformed small lymphocytic lymphoma met criteria for complete response, whereas another met the criteria for partial response (PR). One patient with refractory HD had a PR, whereas three patients had stable disease for up to 9 mo. Conclusion: These results suggest that SAHA has activity in hematol. malignancies including HD and select subtypes of non-Hodgkin's lymphoma.

IT 149647-78-9, Suberoylanilide hydroxamic acid

RL: ADV (Adverse effect, including toxicity); PAC (Pharmacological activity); PKT (Pharmacokinetics); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(oral HDAC inhibitor SAHA showed clin. activity, with toxicities like fatigue, diarrhea, anorexia, dehydration, while IV formulation showed myelosuppression, fatigue, infection, thrombocytopenia in hematol. malignancies patient)

RN 149647-78-9 CAPLUS

CN Octanediamide, N-hydroxy-N'-phenyl- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 4 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:177856 CAPLUS Full-text

DOCUMENT NUMBER: 142:254579

TITLE: Method of treating cancer with histone deacetylase (HDAC) inhibitors

INVENTOR(S): Bacopoulos, Nicholas G.; Chiao, Judy H.; Miller, Thomas A.; Paradise, Carolyn M.; Richon, Victoria M.

PATENT ASSIGNEE(S): Aton Pharma, Inc., USA; Sloan-Kettering Institute for Cancer Research

SOURCE: PCT Int. Appl., 107 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 7

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005018578	A2	20050303	WO 2004-US27943	20040826
WO 2005018578	A3	20050512		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2004087631	A1	20040506	US 2003-650025	20030826
US 2004127523	A1	20040701	US 2003-665079	20030916
AU 2004266169	A1	20050303	AU 2004-266169	20040826
AU 2004266169	A2	20050303		
CA 2535806	AA	20050303	CA 2004-2535806	20040826
EP 1663194	A2	20060607	EP 2004-782425	20040826
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK				
NO 2006001348	A	20060523	NO 2006-1348	20060324

PRIORITY APPLN. INFO.:

US 2003-650025	A1	20030826
US 2003-655079	A	20030916
US 2003-665079	A1	20030916
US 2002-361759P	P	20020304
US 2003-379149	A2	20030304
WO 2004-US27943	W	20040826

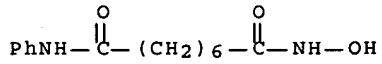
OTHER SOURCE(S): MARPAT 142:254579

AB The invention discloses methods for treating cancers, e.g. mesothelioma or lymphoma. More specifically, the invention discloses methods for treating mesothelioma or diffuse large B-cell lymphoma (DLBCL), by administration of pharmaceutical compns. comprising HDAC inhibitors, e.g., suberoylanilide hydroxamic acid (SAHA; preparation described). The oral formulations of the pharmaceutical compns. have favorable pharmacokinetic profiles such as high bioavailability and surprisingly give rise to high blood levels of the active compds. over an extended period of time. The invention further provides a safe, daily dosing regimen of these pharmaceutical compns., which is easy to follow, and which results in a therapeutically effective amount of the HDAC inhibitors in vivo.

IT 149647-78-9P, Saha
RL: ADV (Adverse effect, including toxicity); PAC (Pharmacological activity); PKT (Pharmacokinetics); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(histone deacetylase inhibitors for cancer treatment)

RN 149647-78-9 CAPLUS

CN Octanediamide, N-hydroxy-N'-phenyl- (9CI) (CA INDEX NAME)



L5 ANSWER 5 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:69409 CAPLUS Full-text

DOCUMENT NUMBER: 142:385256

TITLE: Histone deacetylase inhibitors profoundly decrease proliferation of human lymphoid cancer cell lines

AUTHOR(S): Sakajiri, Sakura; Kumagai, Takashi; Kawamata, Norihiko; Saitoh, Takayuki; Said, Jonathan W.; Koeffler, H. Phillip

CORPORATE SOURCE: Division of Hematology/Oncology, Cedars Sinai Medical Center, UCLA School of Medicine, Los Angeles, CA, USA

SOURCE: Experimental Hematology (New York, NY, United States) (2005), 33(1), 53-61
CODEN: EXHMA6; ISSN: 0301-472X

PUBLISHER: Elsevier Inc.

DOCUMENT TYPE: Journal

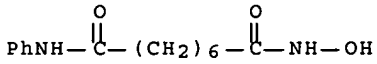
LANGUAGE: English

AB Methylation of tumor suppressor genes is frequently observed in human cancers. These genes are silenced by histone deacetylase (HDAC) recruited by methylated DNA in their promoter regions. HDAC removes acetyl groups from histones and prevents the basic transcriptional machinery access to the target gene, leading to transcriptional repression. HDAC inhibitors (HDACIs) can restore the expression of the tumor suppressor and/or cell cycle regulatory genes in cancer cells and block the cellular proliferation of these cells. In this study, we investigated the in vitro antiproliferative activities of the HDACIs, suberoylanilide hydroxamic acid (SAHA), and valproic acid against 14

human lymphoid cancer cell lines. All of these cell lines were sensitive to the antiproliferative effects of the HDACI. SAHA induced either G1 or G2-M arrest as well as apoptosis. SAHA downregulated cyclin D1 and D2, and upregulated p53, p21, and p27. Chromatin immunopptn. anal. revealed a remarkable increase in the level of acetylated histones associated with the p21 promoter after SAHA treatment. In nude mice, SAHA significantly inhibited growth of a mantle cell lymphoma without major toxic side effects. In summary, HDACIs are promising therapeutic agents for human lymphoid cancers.

IT 149647-78-9, Suberoylanilide hydroxamic acid
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(SAHA showed antiproliferative effect, induced cell cycle arrest, apoptosis, reduced cycline D1, D2, raised p53, p21, p27 in human lymphoid cancer cell lines, showed antitumor activity in NCEBI human mantle lymphoma cell transplanted mouse)

RN 149647-78-9 CAPLUS
CN Octanediamide, N-hydroxy-N'-phenyl- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 6 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:533980 CAPLUS Full-text
DOCUMENT NUMBER: 141:65091
TITLE: Methods of treating cancer with histone deacetylase (HDAC) inhibitors
INVENTOR(S): Bacopoulos, Nicholas G.; Chiao, Judy H.; Miller, Thomas A.; Paradise, Carolyn M.; Richon, Victoria M.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 54 pp., Cont.-in-part of U.S. Ser. No. 379,149.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 7
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004127523	A1	20040701	US 2003-665079	20030916
US 2004072735	A1	20040415	US 2003-379149	20030304
AU 2004266169	A1	20050303	AU 2004-266169	20040826
AU 2004266169	A2	20050303		
CA 2535806	AA	20050303	CA 2004-2535806	20040826
WO 2005018578	A2	20050303	WO 2004-US27943	20040826
WO 2005018578	A3	20050512		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,

AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1663194 A2 20060607 EP 2004-782425 20040826
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK

NO 2006001348 A 20060523 NO 2006-1348 20060324

PRIORITY APPLN. INFO.: US 2002-361759P P 20020304
US 2003-379149 A2 20030304
US 2003-650025 A 20030826
US 2003-655079 A 20030916
US 2003-665079 A 20030916
WO 2004-US27943 W 20040826

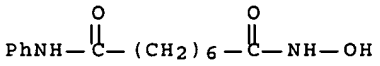
OTHER SOURCE(S): MARPAT 141:65091

AB The invention relates to methods of treating cancers, e.g., lymphoma. More specifically, the present invention relates to methods of treating diffuse large B-cell lymphoma (DLBCL), by administration of pharmaceutical compns. comprising HDAC inhibitors, e.g., suberoylanilide hydroxamic acid (preparation described). The oral formulations of the pharmaceutical compns. have favorable pharmacokinetic profiles such as high bioavailability and surprisingly give rise to high blood levels of the active compds. over an extended period of time. The present invention further provides a safe, daily dosing regimen of these pharmaceutical compns., which is easy to follow, and which results in a therapeutically effective amount of the HDAC inhibitors in vivo.

IT 149647-78-9P, SAHA
RL: PAC (Pharmacological activity); PKT (Pharmacokinetics); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(histone deacetylase inhibitors for treatment of cancer)

RN 149647-78-9 CAPLUS

CN Octanediamide, N-hydroxy-N'-phenyl- (9CI) (CA INDEX NAME)



L5 ANSWER 7 OF 10 USPATFULL on STN

ACCESSION NUMBER: 2006:68089 USPATFULL Full-text

TITLE: Combinations for the treatment of diseases involving cell proliferation

INVENTOR(S): Munzert, Gerd, Ulm, GERMANY, FEDERAL REPUBLIC OF
Steegmaier, Martin, Wien, AUSTRIA
Baum, Anke, Vienna, AUSTRIA

PATENT ASSIGNEE(S): Boehringer Ingelheim International GmbH, Ingelheim, GERMANY, FEDERAL REPUBLIC OF (non-U.S. corporation)

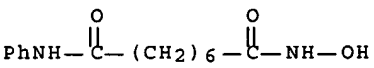
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2006058311	A1	20060316	
APPLICATION INFO.:	US 2005-189540	A1	20050726	(11)

	NUMBER	DATE
PRIORITY INFORMATION:	EP 2004-19361	20040814

EP 2004-19448 20040817
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MICHAEL P. MORRIS, BOEHRINGER INGELHEIM CORPORATION, 900 RIDGEBURY ROAD, P. O. BOX 368, RIDGEFIELD, CT, 06877-0368, US
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 8 Drawing Page(s)
LINE COUNT: 3176
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Disclosed are pharmaceutical compositions for the treatment of diseases which involve cell proliferation. Also disclosed are methods for the treatment of said diseases, comprising co-administration of a compound 1 of Formula (I) ##STR1## wherein the groups L, R.sup.1, R.sup.2, R.sup.3, R.sup.4 and R.sup.5 have the meanings given herein and of an effective amount of an active compound 2 and/or co-treatment with radiation therapy, in a ratio which provides an additive and synergistic effect, and to the combined use of a compound 1 of Formula (I) and of an effective amount of an active compound 2 and/or radiotherapy for the manufacture of corresponding pharmaceutical combination preparations.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
IT 149647-78-9, SAHA
(preparation of aminopteridinones for use in combination therapy for treatment of cell proliferative diseases)
RN 149647-78-9 USPATFULL
CN Octanediamide, N-hydroxy-N'-phenyl- (9CI) (CA INDEX NAME)



L5 ANSWER 8 OF 10 USPATFULL on STN
ACCESSION NUMBER: 2005:331242 USPATFULL Full-text
TITLE: Use of thioredoxin measurements for diagnostics and treatments
INVENTOR(S): Marks, Paul A., Washington, CT, UNITED STATES
Ungerstedt, Johanna, Stockholm, SWEDEN

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005288227	A1	20051229
APPLICATION INFO.:	US 2005-144301	A1	20050603 (11)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2003-369094, filed on 14 Feb 2003, ABANDONED

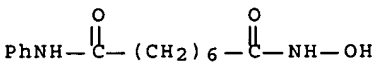
	NUMBER	DATE
PRIORITY INFORMATION:	US 2004-577089P	20040604 (60)
	US 2002-357383P	20020215 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MINTZ LEVIN COHN FERRIS GLOVSKY & POPEO, 666 THIRD AVENUE, NEW YORK, NY, 10017, US

NUMBER OF CLAIMS: 27
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 9 Drawing Page(s)
LINE COUNT: 2218
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention relates to methods for monitoring patient response to histone deacetylase inhibitors (e.g., suberoylanilide hydroxamic acid (SAHA)) or other therapeutic agents by measuring the level of thioredoxin in body fluids, tissues, and/or cells, such as peripheral blood mononuclear cells, plasma, or serum. The invention also relates to methods of monitoring and/or assisting with the diagnosis of a wide variety of thioredoxin-related diseases and conditions, such as inflammatory diseases, allergic diseases, autoimmune diseases, diseases associated with oxidative stress or diseases characterized by cellular hyperproliferation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
IT 149647-78-9, Suberoylanilide hydroxamic acid
(use of thioredoxin expression measurements for diagnostics and monitoring treatments with histone deacetylase inhibitors and other therapeutic agents for hyperproliferative diseases)
RN 149647-78-9 USPATFULL
CN Octanediamide, N-hydroxy-N'-phenyl- (9CI) (CA INDEX NAME)



L5 ANSWER 9 OF 10 USPATFULL on STN
ACCESSION NUMBER: 2004:166081 USPATFULL Full-text
TITLE: Method of Treating Leukemia with a Combination of Suberoylanilide Hydromaxic Acid and Imatinib Mesylate
INVENTOR(S): Bhalla, Kapil N., 12902 Magnolia Drive, MRC-3E 3056D, Tampa, FL, UNITED STATES 33612
Nimmanapalli, Ramadevi, 12902 Magnolia Drive, MRC-3E 3056D, Tampa, FL, UNITED STATES 33612
PATENT ASSIGNEE(S): UNIVERSITY OF SOUTH FLORIDA, Tampa, FL, UNITED STATES (U.S. corporation)

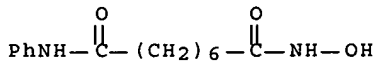
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004127571	A1	20040701	
APPLICATION INFO.:	US 2003-605283	A1	20030919	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-319563P	20020919 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SMITH & HOPEN PA, 15950 BAY VISTA DRIVE, SUITE 220, CLEARWATER, FL, 33760
NUMBER OF CLAIMS: 16
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Page(s)
LINE COUNT: 235
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A method for inducing apoptosis, or increasing the rate or extent of apoptosis, in target cells. The method comprises the steps of contacting the cancer cells with an apoptosis-inducing amount of a tyrosine kinase inhibitor, imatinib mesylate, and a histone deacetylase inhibitor, Suberoylanilide Hydromaxic Acid (SAHA). The method is applicable to ameliorating the resistance of the accelerated and blast phases of CML (CML-BC) to imatinib mesylate.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
IT 149647-78-9, Suberoylanilide hydroxamic acid
(suberoylanilide hydroxamic acid-imatinib mesylate combination for leukemia treatment)
RN 149647-78-9 USPATFULL
CN Octanediamide, N-hydroxy-N'-phenyl- (9CI) (CA INDEX NAME)



L5 ANSWER 10 OF 10 USPATFULL on STN
ACCESSION NUMBER: 2004:166033 USPATFULL Full-text
TITLE: Methods of treating cancer with HDAC inhibitors
INVENTOR(S): Bacopoulos, Nicholas G., New York, NY, UNITED STATES
Chiao, Judy H., Berkeley Heights, NJ, UNITED STATES
Miller, Thomas A., New York, NY, UNITED STATES
Paradise, Carolyn M., Cortland Manor, NY, UNITED STATES
Richon, Victoria M., Rye, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004127523	A1	20040701
APPLICATION INFO.:	US 2003-665079	A1	20030916 (10)

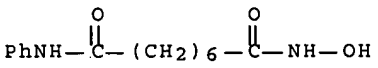
RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2003-379149, filed on 4 Mar 2003, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-361759P	20020304 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MINTZ LEVIN COHN FERRIS GLOVSKY & POPEO, 666 THIRD AVENUE, NEW YORK, NY, 10017
NUMBER OF CLAIMS: 62
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 18 Drawing Page(s)
LINE COUNT: 2792
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to methods of treating cancers, e.g., lymphoma. More specifically, the present invention relates to methods of treating diffuse large B-cell lymphoma (DLBCL), by administration of pharmaceutical compositions comprising HDAC inhibitors, e.g., suberoylanilide hydroxamic acid (SAHA). The oral formulations of the pharmaceutical compositions have favorable pharmacokinetic profiles such as high bioavailability and surprisingly give rise to high blood levels of the active compounds over an extended period of time. The present invention

further provides a safe, daily dosing regimen of these pharmaceutical compositions, which is easy to follow, and which results in a therapeutically effective amount of the HDAC inhibitors in vivo.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
IT 149647-78-9P, SAHA
(histone deacetylase inhibitors for treatment of cancer)
RN 149647-78-9 USPATFULL
CN Octanediamide, N-hydroxy-N'-phenyl- (9CI) (CA INDEX NAME)



=> **d his**

(FILE 'HOME' ENTERED AT 12:18:16 ON 01 DEC 2006)

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L1 STRUCTURE UPLOADED
L2 5 S L1
L3 2 S L1 EXA FULL

FILE 'MEDLINE, CAPLUS, USPATFULL, WPIDS' ENTERED AT 12:19:19 ON 01 DEC 2006
L4 378 S L3
L5 10 S L4 AND "B-CELL LYMPHOMA"

=> **s l4 not py>2003**
L6 81 L4 NOT PY>2003

=> **s l6 and "oral administration"**
L7 8 L6 AND "ORAL ADMINISTRATION"

=> **d l7 1-8 ibib, abs, hitstr**

L7 ANSWER 1 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:405492 CAPLUS Full-text
DOCUMENT NUMBER: 139:115446
TITLE: Molecular sequelae of histone deacetylase inhibition in human malignant B cells
AUTHOR(S): Mitsiades, Nicholas; Mitsiades, Constantine S.; Richardson, Paul G.; McMullan, Ciaran; Poulaki, Vassiliki; Fanourakis, Galinos; Schlossman, Robert; Chauhan, Dharminder; Munshi, Nikhil C.; Hideshima, Teru; Richon, Victoria M.; Marks, Paul A.; Anderson, Kenneth C.
CORPORATE SOURCE: Jerome Lipper Multiple Myeloma Center, Department of Medical Oncology, Dana-Farber Cancer Institute, Harvard Medical School, Boston, MA, USA
SOURCE: Blood (2003), 101(10), 4055-4062
CODEN: BLOOAW; ISSN: 0006-4971
PUBLISHER: American Society of Hematology
DOCUMENT TYPE: Journal

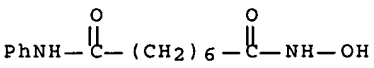
LANGUAGE: English

AB Histone acetylation modulates gene expression, cellular differentiation, and survival and is regulated by the opposing activities of histone acetyltransferases (HATs) and histone deacetylases (HDACs). HDAC inhibition results in accumulation of acetylated nucleosomal histones and induces differentiation and/or apoptosis in transformed cells. In this study, we characterized the effect of suberoylanilide hydroxamic acid (SAHA), the prototype of a series of hydroxamic acid-based HDAC inhibitors, in cell lines and patient cells from B-cell malignancies, including multiple myeloma (MM) and related disorders. SAHA induced apoptosis in all tumor cells tested, with increased p21 and p53 protein levels and dephosphorylation of Rb. We also detected cleavage of Bid, suggesting a role for Bcl-2 family members in regulation of SAHA-induced cell death. Transfection of Bcl-2 cDNA into MM.1S cells completely abrogated SAHA-induced apoptosis, confirming its protective role. SAHA did not induce cleavage of caspase-8, -9, or -3 in MM.1S cells during the early phase of apoptosis, and the pan-caspase inhibitor ZVAD-FMK did not protect against SAHA. Conversely, poly(ADP)ribose polymerase (PARP) was cleaved in a pattern indicative of calpain activation, and the calpain inhibitor calpeptin abrogated SAHA-induced cell death. Importantly, SAHA sensitized MM.1S cells to death receptor-mediated apoptosis and inhibited the secretion of interleukin 6 (IL-6) induced in bone marrow stromal cells (BMSCs) by binding of MM cells, suggesting that it can overcome cell adhesion-mediated drug resistance. Our studies delineate the mechanisms whereby HDAC inhibitors mediate anti-MM activity and overcome drug resistance in the BM milieu and provide the framework for clin. evaluation of SAHA, which is bioavailable, well tolerated, and bioactive after oral administration, to improve patient outcome.

IT 149647-78-9, Suberoylanilide hydroxamic acid
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(histone deacetylase inhibitor; authors characterized the effect of suberoylanilide hydroxamic acid (SAHA) in cell lines and patient cells from B-cell malignancies, including multiple myeloma (MM) and related disorders)

RN 149647-78-9 CAPLUS

CN Octanediamide, N-hydroxy-N'-phenyl- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2003:155835 CAPLUS Full-text

DOCUMENT NUMBER: 139:63206

TITLE: Suberoylanilide hydroxamic acid, a histone deacetylase inhibitor, ameliorates motor deficits in a mouse model of Huntington's disease

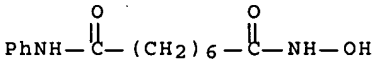
AUTHOR(S): Hockly, Emma; Richon, Victoria M.; Woodman, Benjamin; Smith, Donna L.; Zhou, Xianbo; Rosa, Eddie; Sathasivam, Kirupa; Ghazi-Noori, Shabnam; Mahal, Amarbirpal; Lowden, Philip A. S.; Steffan, Joan S.; Marsh, J. Lawrence; Thompson, Leslie M.; Lewis, Cathryn M.; Marks, Paul A.; Bates, Gillian P.

CORPORATE SOURCE: Medical and Molecular Genetics, Guy's, King's and St. Thomas' School of Medicine, King's College London,

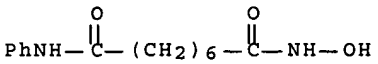
Guy's Hospital, London, SE1 9RT, UK
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (2003), 100(4), 2041-2046
CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Huntington's disease (HD) is an inherited, progressive neurol. disorder that is caused by a CAG/polyglutamine repeat expansion and for which there is no effective therapy. Recent evidence indicates that transcriptional dysregulation may contribute to the mol. pathogenesis of this disease. Supporting this view, administration of histone deacetylase (HDAC) inhibitors has been shown to rescue lethality and photoreceptor neurodegeneration in a Drosophila model of polyglutamine disease. To further explore the therapeutic potential of HDAC inhibitors, we have conducted preclin. trials with suberoylanilide hydroxamic acid (SAHA), a potent HDAC inhibitor, in the R6/2 HD mouse model. We show that SAHA crosses the blood-brain barrier and increases histone acetylation in the brain. We found that SAHA could be administered orally in drinking water when complexed with cyclodextrins. SAHA dramatically improved the motor impairment in R6/2 mice, clearly validating the pursuit of this class of compds. as HD therapeutics.

IT 149647-78-9, Suberoylanilide hydroxamic acid
RL: PAC (Pharmacological activity); PKT (Pharmacokinetics); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(suberoylanilide hydroxamic acid, histone deacetylase inhibitor, ameliorates motor deficits in mouse model of Huntington's disease)
RN 149647-78-9 CAPLUS
CN Octanediamide, N-hydroxy-N'-phenyl- (9CI) (CA INDEX NAME)



IT 149647-78-9D, Suberoylanilide hydroxamic acid, complex with cyclodextrin
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(suberoylanilide hydroxamic acid-cyclodextrin complex as an aqueous solution
for oral administration in the treatment of Huntington's disease)
RN 149647-78-9 CAPLUS
CN Octanediamide, N-hydroxy-N'-phenyl- (9CI) (CA INDEX NAME)



RÉFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:224891 CAPLUS Full-text
DOCUMENT NUMBER: 137:72839
TITLE: The antitumor histone deacetylase inhibitor

suberoylanilide hydroxamic acid exhibits antiinflammatory properties via suppression of cytokines

AUTHOR(S): Leoni, Flavio; Zaliani, Andrea; Bertolini, Giorgio; Porro, Giulia; Pagani, Paolo; Pozzi, Pietro; Dona, Giancarlo; Fossati, Gianluca; Sozzani, Silvano; Azam, Tania; Bufler, Philip; Fantuzzi, Giamila; Goncharov, Igor; Kim, Soo-Hyun; Pomerantz, Benjamin J.; Reznikov, Leonid L.; Siegmund, Britta; Dinarello, Charles A.; Mascagni, Paolo

CORPORATE SOURCE: Italfarmaco, SpA., Balsamo, 20092, Italy

SOURCE: Proceedings of the National Academy of Sciences of the United States of America (2002), 99(5), 2995-3000
CODEN: PNASA6; ISSN: 0027-8424

PUBLISHER: National Academy of Sciences

DOCUMENT TYPE: Journal

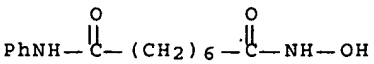
LANGUAGE: English

AB Suberoylanilide hydroxamic acid (SAHA) is a hydroxamic acid-containing hybrid polar mol.; SAHA specifically binds to and inhibits the activity of histone deacetylase. Although SAHA, like other inhibitors of histone deacetylase, exhibits antitumor effects by increasing expression of genes regulating tumor survival, we found that SAHA reduces the production of proinflammatory cytokines in vivo and in vitro. A single oral administration of SAHA to mice dose-dependently reduced circulating TNF-α, IL-1β, IL-6, and IFN-γ induced by lipopolysaccharide (LPS). Administration of SAHA also reduced hepatic cellular injury in mice following i.v. injection of Con A. SAHA inhibited nitric oxide release in mouse macrophages stimulated by the combination of TNF-α plus IFN-γ. Human peripheral blood mononuclear cells stimulated with LPS in the presence of SAHA released less TNF-α, IL-1β, IL-12, and IFN-γ (50% reduction at 100-200 nM). The production of IFN-γ stimulated by IL-18 plus IL-12 was also inhibited by SAHA (85% at 200 nM). However, SAHA did not affect LPS-induced synthesis of the IL-1β precursor, the IL-1 receptor antagonist, or the chemokine IL-8. In addition, IFN-γ induced by anti-CD3 was not suppressed by SAHA. Steady-state mRNA levels for LPS-induced TNF-α and IFN-γ in peripheral blood mononuclear cells were markedly decreased, whereas IL-8 and IL-1β mRNA levels were unaffected. Because SAHA exhibits antiinflammatory properties in vivo and in vitro, inhibitors of histone deacetylase may stimulate the expression of genes that control the synthesis of cytokines and nitric oxide or hyper-acetylate other targets.

IT 149647-78-9
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(antitumor histone deacetylase inhibitor suberoylanilide hydroxamic acid exhibits antiinflammatory properties via suppression of cytokines)

RN 149647-78-9 CAPLUS

CN Octanediamide, N-hydroxy-N'-phenyl- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 4 OF 8 USPATFULL on STN

ACCESSION NUMBER: 2003:334712 USPATFULL Full-text
TITLE: Method of treating TRX mediated diseases
INVENTOR(S): Richon, Victoria M., Rye, NY, UNITED STATES
Marks, Paul A., Washington, CT, UNITED STATES
Rifkind, Richard A., New York, NY, UNITED STATES
Butler, Lisa M., Athelstone, AUSTRALIA

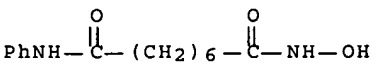
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003235588	A1	20031225
APPLICATION INFO.:	US 2003-369094	A1	20030214 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-357383P	20020215 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HAMILTON, BROOK, SMITH & REYNOLDS, P.C., 530 VIRGINIA ROAD, P.O. BOX 9133, CONCORD, MA, 01742-9133
NUMBER OF CLAIMS: 90
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 9 Drawing Page(s)
LINE COUNT: 2095
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides a novel method for treating and/or preventing thioredoxin (TRX)-mediated diseases and conditions, by administering to a subject in need of such treatment a therapeutically effective amount of a histone deacetylase (HDAC) inhibitor or a pharmaceutically acceptable salt or hydrate thereof. The HDAC inhibitor can alter the expression of a thioredoxin-binding-protein (e.g. TBP-2), which in turn can lead to an altered TRX/thioredoxin-binding-protein cellular binding interaction, resulting in an increase or decrease in the level or activity of cellular TRX, for example the expression level or reducing activity of TRX. Thus the present invention relates to the use of HDAC inhibitors in a method of preventing and/or treating a wide variety of thioredoxin (TRX)-mediated diseases and conditions, such as inflammatory diseases, allergic diseases, autoimmune diseases, diseases associated with oxidative stress or diseases characterized by cellular hyperproliferation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
IT 149647-78-9, SAHA
(use of histone deacetylase inhibitors for preventing/treating thioredoxin (TRX) mediated diseases or conditions associated with inflammation and cellular hyperproliferation)
RN 149647-78-9 USPATFULL
CN Octanediamide, N-hydroxy-N'-phenyl- (9CI) (CA INDEX NAME)



L7 ANSWER 5 OF 8 USPATFULL on STN
ACCESSION NUMBER: 2003:166667 USPATFULL Full-text
TITLE: Method of treating autoimmune diseases
INVENTOR(S): Kammer, Gary M., Lewisville, NC, UNITED STATES

Mishra, Nilamadhab, Winston-Salem, NC, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003114525	A1	20030619
APPLICATION INFO.:	US 2002-151481	A1	20020520 (10)

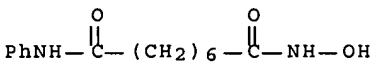
RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-718195, filed on 20 Nov 2000, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	WO 2001-US43871	20011119

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MYERS BIGEL SIBLEY & SAJOVEC, PO BOX 37428, RALEIGH, NC, 27627
NUMBER OF CLAIMS: 36
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 25 Drawing Page(s)
LINE COUNT: 1464
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A method of treating an autoimmune disease comprising administering to the subject a treatment effective amount of a histone hyperacetylating agent, or a pharmaceutically acceptable salt thereof.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
IT 149647-78-9, Suberoylanilide Hydroxamic acid
(method of treating autoimmune diseases using a histone hyperacetylating agent)
RN 149647-78-9 USPATFULL
CN Octanediamide, N-hydroxy-N'-phenyl- (9CI) (CA INDEX NAME)



L7 ANSWER 6 OF 8 USPATFULL on STN
ACCESSION NUMBER: 2003:140935 USPATFULL Full-text
TITLE: Modulation of gene expression by combination therapy
INVENTOR(S): Besterman, Jeffrey M., Bai D'Urfe, CANADA
MacLeod, Robert A., Westmount, CANADA
Siders, William M., Watertown, MA, UNITED STATES
Li, Zuomei, Kirkland, CANADA

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003096777	A1	20030522
APPLICATION INFO.:	US 2002-145493	A1	20020514 (10)

RELATED APPLN. INFO.: Division of Ser. No. US 1999-420692, filed on 19 Oct 1999, PENDING

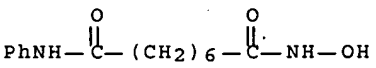
	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-104804P	19981019 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: KEOWN & ASSOCIATES, 500 WEST CUMMINGS PARK, SUITE 1200, WOBURN, MA, 01801
NUMBER OF CLAIMS: 50
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 31 Drawing Page(s)
LINE COUNT: 2704
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to the modulation of gene expression. In particular, the invention relates to compositions comprising antisense oligonucleotides which inhibit expression of a gene in operable association with protein effectors of a product of that gene, and methods of using the same.

In addition, the invention relates to the modulation of mammalian gene expression regulated by methylation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
IT 149647-78-9
(antisense oligonucleotide and gene product protein effector for gene expression modulation)
RN 149647-78-9 USPATFULL
CN Octanediamide, N-hydroxy-N'-phenyl- (9CI) (CA INDEX NAME)

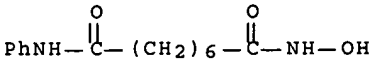


L7 ANSWER 7 OF 8 USPATFULL on STN
ACCESSION NUMBER: 2003:120185 USPATFULL Full-text
TITLE: Method of treating autoimmune diseases
INVENTOR(S): Kammer, Gary M., Lewisville, NC, UNITED STATES
Mishra, Nilamadhab, Winston-Salem, NC, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003082666	A1	20030501
APPLICATION INFO.:	US 2002-187586	A1	20020702 (10)

RELATED APPLN. INFO.: Division of Ser. No. US 2000-718195, filed on 20 Nov 2000, ABANDONED
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MYERS BIGEL SIBLEY & SAJOVEC, PO BOX 37428, RALEIGH, NC, 27627
NUMBER OF CLAIMS: 29
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Page(s)
LINE COUNT: 843
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of treating an autoimmune disease (for example, Systemic Lupus Erythematosus) comprises administering to the subject a treatment effective amount of a histone hyperacetylating agent, or a pharmaceutically acceptable salt thereof. Methods of screening compounds useful for the treatment of autoimmune disease are also disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
IT 149647-78-9
(method of treating autoimmune diseases with histone hyperacetylating agent)
RN 149647-78-9 USPATFULL
CN Octanediamide, N-hydroxy-N'-phenyl- (9CI) (CA INDEX NAME)



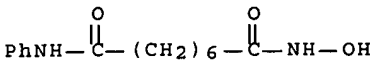
L7 ANSWER 8 OF 8 USPATFULL on STN

ACCESSION NUMBER:	2002:337369 USPATFULL Full-text		
TITLE:	MAP-2 as a determinant of metastatic potential		
INVENTOR(S):	Setaluri, Vijayasaradhi, Winston-Salem, NC, UNITED STATES Fang, Dong, Winston-Salem, NC, UNITED STATES White, Wain, Winston-Salem, NC, UNITED STATES		
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002192727	A1	20021219
	US 6613534	B2	20030902
APPLICATION INFO.:	US 2001-812348	A1	20010320 (9)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	KILPATRICK STOCKTON LLP, 1001 WEST FOURTH STREET, WINSTON-SALEM, NC, 27101		
NUMBER OF CLAIMS:	68		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	8 Drawing Page(s)		
LINE COUNT:	1730		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention relates to detection of MAP-2 (microtubule associated protein-2) as a marker to determine the metastatic potential of a tumor, including tumors derived from the neural crest such as melanomas, gliomas, Schwanomas, chromocytomas and small cell lung cancer. In one aspect, the invention comprises a method for determining the metastatic potential of a tumor sample, wherein decreased levels of MAP-2 expression in a test sample relative to controls indicates that the sample has increased metastatic potential as compared to the control. In another aspect, the invention comprises a method to prevent tumor progression in metastatic melanoma by increasing levels of MAP-2 protein in cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
IT 149647-78-9
(microtubule associated protein-2 (MAP-2) and its role as an indicator of metastatic potential)
RN 149647-78-9 USPATFULL
CN Octanediamide, N-hydroxy-N'-phenyl- (9CI) (CA INDEX NAME)



=> **d his**

(FILE 'HOME' ENTERED AT 12:18:16 ON 01 DEC 2006)

FILE 'REGISTRY' ENTERED AT 12:18:29 ON 01 DEC 2006
L1 STRUCTURE UPLOADED
L2 5 S L1
L3 2 S L1 EXA FULL

FILE 'MEDLINE, CAPLUS, USPATFULL, WPIDS' ENTERED AT 12:19:19 ON 01 DEC 2006
L4 378 S L3
L5 10 S L4 AND "B-CELL LYMPHOMA"
L6 81 S L4 NOT PY>2003
L7 8 S L6 AND "ORAL ADMINISTRATION"

=> **s l6 and cancer**
L8 36 L6 AND CANCER

=> **s l6 and lymphoma**
L9 2 L6 AND LYMPHOMA

=> **d l9 1-2 ibib, abs**

L9 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:847686 CAPLUS Full-text
DOCUMENT NUMBER: 139:358271
TITLE: Phase I clinical trial of histone deacetylase inhibitor: suberoylanilide hydroxamic acid administered intravenously
AUTHOR(S): Kelly, Wm. Kevin; Richon, Victoria M.; O'Connor, Owen; Curley, Tracy; MacGregor-Curtelli, Barbara; Tong, William; Klang, Mark; Schwartz, Lawrence; Richardson, Stacie; Rosa, Eddie; Drobnjak, Marija; Cordon-Cordo, Carlos; Chiao, Judy H.; Rifkind, Richard; Marks, Paul A.; Scher, Howard
CORPORATE SOURCE: Genitourinary Oncology Service, Department of Medicine, Memorial Sloan-Kettering Cancer Center and Joan and Sanford Weill Medical College of Cornell University, New York, NY, 10021, USA
SOURCE: Clinical Cancer Research (2003), 9(10, Pt. 1), 3578-3588
CODEN: CCREF4; ISSN: 1078-0432
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English
AB To evaluate the safety, pharmacokinetics, and biol. activity of suberoylanilide hydroxamic acid (SAHA) administered by 2-h i.v. infusion in patients with advanced cancer. SAHA was administered for 3 days every 21 days in part A and 5 days for 1-3 wk in part B. Dose escalation proceeded independently in patients with solid tumor and hematol. malignancies (part B only). Pharmacokinetic studies were performed along with assessment of acetylated histones in peripheral blood mononuclear cells and tumor tissues. No dose-limiting toxicities were observed in 8 patients enrolled in part A

(75, 150, 300, 600, and 900 mg/m2/day). Among 12 hematol. and 17 solid tumor patients enrolled in part B (300, 600, and 900 mg/m2/day), therapy was delayed ≥1 wk for grade 3/4 leukopenia and/or thrombocytopenia in 2 of 5 hematol. patients at 600 mg/m2/day + 5 days for 3 wk. The maximal-tolerated dose was 300 mg/m2/day + 5 days for 3 wk for hematol. patients. One solid patient on 900 mg/m2/day + 5 days for 3 wk developed acute respiratory distress and grade 3 hypotension. The cohort was expanded to 6 patients, and no addnl. dose-limiting toxicities were observed Mean terminal half-life ranged from 21 to 58 min, and there, was dose-proportional increase in area under the curve. An accumulation of acetylated histones in peripheral blood mononuclear cells up to 4 h postinfusion was observed at higher dose levels. Posttherapy tumor biopsies showed an accumulation of acetylated histones by immunohistochem. Four (2 lymphoma and 2 bladder) patients had objective tumor regression with clin. improvement in tumor related symptoms. Daily i.v. SAHA is well tolerated, inhibits the biol. target in vivo, and has antitumor activity in solid and hematol. tumors.

REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 2 OF 2 USPATFULL on STN

ACCESSION NUMBER: 2002:323219 USPATFULL Full-text

TITLE: Use of retinoids plus histone deacetylase inhibitors to inhibit the growth of solid tumors

INVENTOR(S): Gudas, Lorraine J., New York, NY, UNITED STATES
Nanus, David, New Rochelle, NY, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002183388	A1	20021205	
APPLICATION INFO.:	US 2002-61101	A1	20020201	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-265651P	20010201 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Michael L. Goldman, Esq., NIXON PEABODY LLP, Clinton Square, P.O. Box 31051, Rochester, NY, 14603-1051
NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 20 Drawing Page(s)
LINE COUNT: 549
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides a method of inhibiting growth of solid tumors in an animal which comprises administering an effective amount of trichostatin A (TSA) to an animal in need of such treatment. The present invention also provides a method of inhibiting growth of solid tumors in an animal which comprises administering an effective amount of a histone deacetylase inhibitor and a retinoid to an animal in need of such treatment. Examples of solid tumors which may be treated using the methods of the invention include but are not limited to carcinomas of the head and neck, breast, skin, kidney, oral cavity, colon, prostate, pancreas and lung.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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(FILE 'HOME' ENTERED AT 12:18:16 ON 01 DEC 2006)

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     FILE 'REGISTRY' ENTERED AT 12:18:29 ON 01 DEC 2006
L1              STRUCTURE UPLOADED
L2            5 S L1
L3            2 S L1 EXA FULL

     FILE 'MEDLINE, CAPLUS, USPATFULL, WPIDS' ENTERED AT 12:19:19 ON 01 DEC
     2006
L4          378 S L3
L5           10 S L4 AND "B-CELL LYMPHOMA"
L6           81 S L4 NOT PY>2003
L7            8 S L6 AND "ORAL ADMINISTRATION"
L8           36 S L6 AND CANCER
L9            2 S L6 AND LYMPHOMA

=> s l6 and "orally"
L10           7 L6 AND "ORALLY"

=> d l10 1-7 ibib, abs

L10  ANSWER 1 OF 7  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:        2003:155835  CAPLUS  Full-text
DOCUMENT NUMBER:         139:63206
TITLE:                   Suberoylanilide hydroxamic acid, a histone deacetylase
                         inhibitor, ameliorates motor deficits in a mouse model
                         of Huntington's disease
AUTHOR(S):               Hockly, Emma; Richon, Victoria M.; Woodman, Benjamin;
                         Smith, Donna L.; Zhou, Xianbo; Rosa, Eddie;
                         Sathasivam, Kirupa; Ghazi-Noori, Shabnam; Mahal,
                         Amarbirpal; Lowden, Philip A. S.; Steffan, Joan S.;
                         Marsh, J. Lawrence; Thompson, Leslie M.; Lewis,
                         Cathryn M.; Marks, Paul A.; Bates, Gillian P.
CORPORATE SOURCE:        Medical and Molecular Genetics, Guy's, King's and St.
                         Thomas' School of Medicine, King's College London,
                         Guy's Hospital, London, SE1 9RT, UK
SOURCE:                  Proceedings of the National Academy of Sciences of the
                         United States of America (2003), 100(4), 2041-2046
                         CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER:               National Academy of Sciences
DOCUMENT TYPE:           Journal
LANGUAGE:                English
AB    Huntington's disease (HD) is an inherited, progressive neurol. disorder that
      is caused by a CAG/polyglutamine repeat expansion and for which there is no
      effective therapy.  Recent evidence indicates that transcriptional
      dysregulation may contribute to the mol. pathogenesis of this disease.
      Supporting this view, administration of histone deacetylase (HDAC) inhibitors
      has been shown to rescue lethality and photoreceptor neurodegeneration in a
      Drosophila model of polyglutamine disease.  To further explore the therapeutic
      potential of HDAC inhibitors, we have conducted preclin. trials with
      suberoylanilide hydroxamic acid (SAHA), a potent HDAC inhibitor, in the R6/2
      HD mouse model.  We show that SAHA crosses the blood-brain barrier and
      increases histone acetylation in the brain.  We found that SAHA could be
      administered orally in drinking water when complexed with cyclodextrins.  SAHA
      dramatically improved the motor impairment in R6/2 mice, clearly validating
      the pursuit of this class of compds. as HD therapeutics.
REFERENCE COUNT:         36    THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS
                               RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10  ANSWER 2 OF 7  USPATFULL on STN
ACCESSION NUMBER:        2003:334712  USPATFULL  Full-text
```

TITLE: Method of treating TRX mediated diseases
INVENTOR(S): Richon, Victoria M., Rye, NY, UNITED STATES
Marks, Paul A., Washington, CT, UNITED STATES
Rifkind, Richard A., New York, NY, UNITED STATES
Butler, Lisa M., Athelstone, AUSTRALIA

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003235588	A1	20031225
APPLICATION INFO.:	US 2003-369094	A1	20030214 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-357383P	20020215 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HAMILTON, BROOK, SMITH & REYNOLDS, P.C., 530 VIRGINIA ROAD, P.O. BOX 9133, CONCORD, MA, 01742-9133
NUMBER OF CLAIMS: 90
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 9 Drawing Page(s)
LINE COUNT: 2095
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides a novel method for treating and/or preventing thioredoxin (TRX)-mediated diseases and conditions, by administering to a subject in need of such treatment a therapeutically effective amount of a histone deacetylase (HDAC) inhibitor or a pharmaceutically acceptable salt or hydrate thereof. The HDAC inhibitor can alter the expression of a thioredoxin-binding-protein (e.g. TBP-2), which in turn can lead to an altered TRX/thioredoxin-binding-protein cellular binding interaction, resulting in an increase or decrease in the level or activity of cellular TRX, for example the expression level or reducing activity of TRX. Thus the present invention relates to the use of HDAC inhibitors in a method of preventing and/or treating a wide variety of thioredoxin (TRX)-mediated diseases and conditions, such as inflammatory diseases, allergic diseases, autoimmune diseases, diseases associated with oxidative stress or diseases characterized by cellular hyperproliferation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 3 OF 7 USPATFULL on STN
ACCESSION NUMBER: 2002:323219 USPATFULL Full-text
TITLE: Use of retinoids plus histone deacetylase inhibitors to inhibit the growth of solid tumors
INVENTOR(S): Gudas, Lorraine J., New York, NY, UNITED STATES
Nanus, David, New Rochelle, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002183388	A1	20021205
APPLICATION INFO.:	US 2002-61101	A1	20020201 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-265651P	20010201 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Michael L. Goldman, Esq., NIXON PEABODY LLP, Clinton Square, P.O. Box 31051, Rochester, NY, 14603-1051

NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 20 Drawing Page(s)
LINE COUNT: 549
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a method of inhibiting growth of solid tumors in an animal which comprises administering an effective amount of trichostatin A (TSA) to an animal in need of such treatment. The present invention also provides a method of inhibiting growth of solid tumors in an animal which comprises administering an effective amount of a histone deacetylase inhibitor and a retinoid to an animal in need of such treatment. Examples of solid tumors which may be treated using the methods of the invention include but are not limited to carcinomas of the head and neck, breast, skin, kidney, oral cavity, colon, prostate, pancreas and lung.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 4 OF 7 USPATFULL on STN
ACCESSION NUMBER: 2000:88196 USPATFULL Full-text
TITLE: Potent inducers of terminal differentiation and methods of use thereof
INVENTOR(S): Breslow, Ronald, Englewood, NJ, United States
Marks, Paul A., Bridgewater, CT, United States
Rifkind, Richard A., New York, NY, United States
Jursic, Branko, New Orleans, LA, United States
PATENT ASSIGNEE(S): Sloan-Kettering Institute for Cancer Research, New York, NY, United States (U.S. corporation)
The Trustees of Columbia University in the City of New York, New York, NY, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6087367		20000711
APPLICATION INFO.:	US 1999-314195		19990518 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1994-222685, filed on 4 Apr 1994 which is a division of Ser. No. US 1991-771760, filed on 4 Oct 1991, now patented, Pat. No. US 5369108, issued on 29 Nov 1994
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Kight, John
ASSISTANT EXAMINER: Covington, Raymond
LEGAL REPRESENTATIVE: White, John P.Cooper & Dunham LLP
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
LINE COUNT: 1379
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides the compound having the structure: ##STR1## wherein each of R.sub.1 and R.sub.2 are independently the same as or different from each other; when R.sub.1 and R.sub.2 are the same, each is a substituted or unsubstituted arylamino, cycloalkylamino, pyridineamino, piperidino, 9-purine-6-amine, or thiozoleamino group; when R.sub.1 and R.sub.2 are different, R.sub.1 =R.sub.3 --N--R.sub.4, wherein each of R.sub.3 and R.sub.4 are independently the same as or different from each other and are a hydrogen atom, a hydroxyl group, a substituted or unsubstituted, branched or unbranched alkyl, alkenyl, cycloalkyl, aryl, alkyloxy, aryloxy, arylalkyloxy, or pyridine group, or R.sub.3 and R.sub.4 bond together to form a piperidine group and R.sub.2 is a hydroxylamino,

hydroxyl, amino, alkylamino, dialkylamino or alkyloxy group; and n is an integer from about 4 to about 8.

The present invention also provides a method of selectively inducing terminal differentiation of neoplastic cells and thereby inhibiting proliferation of such cells. Moreover, the present invention provides a method of treating a patient having a tumor characterized by proliferation of neoplastic cells. Lastly, the present invention provides a pharmaceutical composition comprising a pharmaceutically acceptable carrier and a therapeutically acceptable amount of the compound above.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 5 OF 7 USPATFULL on STN
ACCESSION NUMBER: 1999:89192 USPATFULL Full-text
TITLE: Potent inducers of terminal differentiation and methods of use thereof
INVENTOR(S): Breslow, Ronald, Englewood, NJ, United States
Marks, Paul A., Bridgewater, CT, United States
Rifkind, Richard A., New York, NY, United States
Jursic, Branko, New Orleans, LA, United States
PATENT ASSIGNEE(S): Sloan-Kettering Institute for Cancer Research, New York, NY, United States (U.S. corporation)
The Trustees of Columbia Univerisity in the City of New York, New York, NY, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5932616		19990803
APPLICATION INFO.:	US 1994-222685		19940404 (8)

RELATED APPLN. INFO.: Division of Ser. No. US 1991-771760, filed on 4 Oct 1991, now patented, Pat. No. US 5369108
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Kight, John
ASSISTANT EXAMINER: Covington, Raymond
LEGAL REPRESENTATIVE: White, John P.Cooper & Dunham LLP
NUMBER OF CLAIMS: 27
EXEMPLARY CLAIM: 1
LINE COUNT: 1418
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides the compound having the structure: ##STR1## wherein each of R.sub.1 and R.sub.2 are independently the same as or different from each other; when R.sub.1 and R.sub.2 are the same, each is a substituted or unsubstituted arylamino, cycloalkylamino, pyridineamino, piperidino, 9-purine-6-amine, or thiozoleamino group; when R.sub.1 and R.sub.2 are different, R.sub.1 =R.sub.3 --N--R.sub.4, wherein each of R.sub.3 and R.sub.4 are independently the same as or different from each other and are a hydrogen atom, a hydroxyl group, a substituted or unsubstituted, branched or unbranched alkyl, alkenyl, cycloalkyl, aryl, alkyloxy, aryloxy, arylalkyloxy, or pyridine group, or R.sub.3 and R.sub.4 bond together to form a piperidine group and R.sub.2 is a hydroxylamino, hydroxyl, amino, alkylamino, dialkylamino or alkyloxy group; and n is an integer from about 4 to about 8.

The present invention also provides a method of selectively inducing terminal differentiation of neoplastic cells and thereby inhibiting

proliferation of such cells. Moreover, the present invention provides a method of treating a patient having a tumor characterized by proliferation of neoplastic cells. Lastly, the present invention provides a pharmaceutical composition comprising a pharmaceutically acceptable carrier and a therapeutically acceptable amount of the compound above.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 6 OF 7 USPATFULL on STN

ACCESSION NUMBER:	97:120628 USPATFULL Full-text
TITLE:	Potent inducers of terminal differentiation and method of use thereof
INVENTOR(S):	Breslow, Ronald, Englewood, NJ, United States Marks, Paul A., Washington, CT, United States Rifkind, Richard A., New York, NY, United States
PATENT ASSIGNEE(S):	Sloan-Kettering Institute for Cancer Research, New York, NY, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5700811		19971223
APPLICATION INFO.:	US 1994-246363		19940519 (8)

RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1991-771760, filed on 4 Oct 1991, now patented, Pat. No. US 5369108
DOCUMENT TYPE:	Utility
FILE SEGMENT:	Granted
PRIMARY EXAMINER:	Ivy, C. Warren
ASSISTANT EXAMINER:	Covington, Raymond
LEGAL REPRESENTATIVE:	White, John P.
NUMBER OF CLAIMS:	18
EXEMPLARY CLAIM:	1
LINE COUNT:	1585

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention is directed to compounds having the structure: ##STR1## wherein R.sub.1 and R.sub.2 are independently the same as or different from each other; when R.sub.1 and R.sub.2 are the same, each is a substituted or unsubstituted arylamino, cycloalkylamino, pyridineamino, piperidino, 9-purine-6-amine or thiazoleamino group; when R.sub.1 and R.sub.2 are different, R.sub.1 =R.sub.3 --N--R.sub.4, and n is an integer from about 4 to about 8. This invention also provides a method of selectively inducing terminal differentiation of neoplastic cells and thereby inhibiting proliferation of such cells. The invention further provides pharmaceutical compositions comprising a therapeutically effective amount of the compounds of the present invention and a pharmaceutically acceptable carrier.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 7 OF 7 USPATFULL on STN

ACCESSION NUMBER:	94:104578 USPATFULL Full-text
TITLE:	Potent inducers of terminal differentiation and methods of use thereof
INVENTOR(S):	Breslow, Ronald, Englewood, NJ, United States Marks, Paul A., Bridgewater, CT, United States Rifkind, Richard A., New York, NY, United States Jursic, Branko, New Orleans, LA, United States
PATENT ASSIGNEE(S):	Sloan-Kettering Institute for Cancer Research, New York, NY, United States (U.S. corporation) The Trustees of Columbia, New York, NY, United States

(U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5369108		19941129	
APPLICATION INFO.:	US 1991-771760		19911004	(7)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	Granted			
PRIMARY EXAMINER:	Ivy, C. Warren			
ASSISTANT EXAMINER:	Covington, Raymond			
LEGAL REPRESENTATIVE:	White, John P.			
NUMBER OF CLAIMS:	17			
EXEMPLARY CLAIM:	1			
LINE COUNT:	1291			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides the compound having the structure: ##STR1## wherein each of R.sub.1 and R.sub.2 are independently the same as or different from each other; when R.sub.1 and R.sub.2 are the same, each is a substituted or unsubstituted arylamino, cycloalkylamino, pyridineamino, piperidino, 9-purine-6-amine, or thiozoleamino group; when R.sub.1 and R.sub.2 are different, R.sub.1 =R.sub.3 --N--R.sub.4, wherein each of R.sub.3 and R.sub.4 are independently the same as or different from each other and are a hydrogen atom, a hydroxyl group, a substituted or unsubstituted, branched or unbranched alkyl, alkenyl, cycloalkyl, aryl, alkyloxy, aryloxy, arylalkyloxy, or pyridine group, or R.sub.3 and R.sub.4 bond together to form a piperidine group and R.sub.2 is a hydroxylamino, hydroxyl, amino, alkylamino, dialkylamino or alkyloxy group; and n is an integer from about 4 to about 8.

The present invention also provides a method of selectively inducing terminal differentiation of neoplastic cells and thereby inhibiting proliferation of such cells. Moreover, the present invention provides a method of treating a patient having a tumor characterized by proliferation of neoplastic cells. Lastly, the present invention provides a pharmaceutical composition comprising a pharmaceutically acceptable carrier and a therapeutically acceptable amount of the compound above.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> **d his**

(FILE 'HOME' ENTERED AT 12:18:16 ON 01 DEC 2006)

FILE 'REGISTRY' ENTERED AT 12:18:29 ON 01 DEC 2006

L1 STRUCTURE UPLOADED
L2 5 S L1
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FILE 'MEDLINE, CAPLUS, USPATFULL, WPIDS' ENTERED AT 12:19:19 ON 01 DEC 2006

L4 378 S L3
L5 10 S L4 AND "B-CELL LYMPHOMA"
L6 81 S L4 NOT PY>2003
L7 8 S L6 AND "ORAL ADMINISTRATION"
L8 36 S L6 AND CANCER
L9 2 S L6 AND LYMPHOMA
L10 7 S L6 AND "ORALLY"

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